ALCOHOL WITHDRAWAL AND HYPOKALAEMIA: A CASE REPORT

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(Received 12 April 1999; in revised form 28 August 1999; accepted 10 September 1999)

Abstract — A case is presented where a 25-year-old man developed a serious hypokalaemia (K⁺ 2.2 mmol/l) during alcohol withdrawal, despite intravenous saline treatment and normal feeding. As hypokalaemia can be symptom-free, we want to draw attention to the combination of vomiting, malnutrition and alcohol withdrawal, as these can cause lethal complications. We therefore recommend that potassium serum level should be routinely monitored during alcohol withdrawal, even when this is being managed in the community.

INTRODUCTION

There are a number of published reports showing that serum potassium concentration falls during alcohol withdrawal, especially if complicated by delirium tremens (Meyer and Urban, 1977; Wadstein and Skude, 1978; Watson et al., 1984; Laso et al., 1990; Carl and Holzbach, 1994; Wetterling et al., 1994). Some studies even show evidence that potassium could be useful as an indicator to predict delirium tremens (Wadstein and Skude, 1978; Wetterling et al., 1994). Others suggest that there is no linear correlation between potassium level and possible development of delirium tremens (Nanji and Blank, 1984).

The case presented here shows that the combination of vomiting, malnutrition and alcohol withdrawal can cause a potentially dangerous drop in serum potassium level.

CASE HISTORY

T.N., a 25-year-old man, well known to our service (an outpatient at the Alcohol Treatment Unit) presented on a Monday afternoon. He has a history of delirium tremens and withdrawal fits and extreme self-neglect while drinking. He had been discharged from hospital that morning after a short in-patient detoxification. He had been admitted early on the previous Friday morning from the Accident and Emergency Department of a local hospital after he fell down the stairs under the influence of alcohol. He had been on a binge for 13 days. He was treated for a supraorbital laceration. When ready for discharge, he started complaining of nausea and vomited twice. As a result, he was admitted for detoxification. A routine blood test revealed a potassium level of 3.1 mmol/l. He was treated with an intravenous saline (0.9% NaCl) infusion (1 l) and 15 mg of diazepam daily (intramuscularly the first day and orally the following 2 days). Except for nausea during the first 2 days, the patient experienced no physical symptoms at all and started eating again. He did not develop delirium tremens. On discharge from hospital on the Monday morning, he received his last dose of diazepam (5 mg) and was symptom-free.

When he contacted our service on the Monday afternoon, he was complaining of weakness and suffered from nausea again; he had not been vomiting since his discharge from hospital. Blood tests were repeated. His potassium level had dropped to 2.2 mmol/l. An urgent referral to the Accident and Emergency Department had to be arranged. He was immediately treated with an intravenous saline infusion (1 l) with 40 mmol potassium chloride in it and intravenous thiamine (Pabrinex: ascorbic acid 500 mg, anhydrous glucose 1 g, nicotinamide 160 mg, pyridoxine hydrochloride 50 mg, riboflavin 4 mg, thiamine hydrochloride 250 mg). Oral diazepam, 10 mg three times daily, was recommenced and oral potassium supplementation was given (Sando-K: potassium 470 mg, chloride 285 mg, four tablets). The next day, his potassium level rose to 2.8 mmol/l. Potassium replacement and thiamine were continued orally (Sando-K, four tablets three times daily, thiamine 100 mg three times daily) and diazepam was reduced to 5 mg three times daily. He was discharged on the Wednesday with a potassium level of 4.3 mmol/l. Potassium supplements were stopped on discharge. No other clinical cause of hypokalaemia was revealed. Continued monitoring of potassium until 1 week after his discharge revealed no abnormalities.

DISCUSSION

Medical records revealed that our patient had also developed a hypokalaemia during a previous detoxification 8 months earlier. On the day of admission his potassium level had been 3.9 mmol/l. On the third day of the admission, his potassium had dropped to 3.2 mmol/l. On day 6, potassium was normalized to 3.9 mmol/l. On that occasion, there had been no history of vomiting preceding his detoxification, and he had not developed delirium tremens.

Wadstein and Skude (1978) described 26 patients in whom a steady decrease in serum potassium led to hypokalaemia when delirium tremens started. Serum potassium returned to normal with recovery from delirium tremens, and potassium remained unchanged in patients who did not develop delirium tremens. Wetterling et al. (1994) also reported that a decreased serum potassium and chloride indicate a higher risk for the development of delirium tremens. Nanji and Blank (1984), however, reported no significant difference in serum potassium between patients with and without delirium tremens.

Carl and Holzbach (1994) found that the more pronounced the alcohol withdrawal, the sharper the decline in the levels of potassium and magnesium. In each of their cases, the decline in serum magnesium level preceded that of potassium level by...
HYPOKALAEMIA IN ALCOHOL WITHDRAWAL 1 day. Laso et al. (1990) also showed a close negative correlation between intensity of withdrawal and serum potassium. Meyer and Urban (1977) revealed a significant decrease of potassium in their group of alcoholics with withdrawal seizures. Serum potassium remained within the normal range (although low) in their control group (withdrawing alcoholics without seizures).

Watson et al. (1984) reported significantly lower total body potassium in alcoholics, compared to non-alcoholics. They found no correlation between total body potassium and day 1 serum potassium levels. However, there was a significant positive correlation between total body potassium and the minimum serum potassium level recorded during the withdrawal period.

The mechanism behind serum electrolyte disturbances during alcohol withdrawal is still poorly understood. As alcoholics are already at higher risk of developing hypokalaemia due to vomiting, diarrhoea and malnutrition whilst drinking, it is clear that during alcohol withdrawal, even in a community setting, careful monitoring of electrolyte concentrations is necessary. This should be conducted, as in the case described, even where symptoms of vomiting and diarrhoea are mild. Further research is needed in order to identify whether or not it would be wise to monitor some or all patients in whom vomiting and diarrhoea are absent. During potassium supplementation, close monitoring is needed to follow normalization of the potassium level and after discontinuation monitoring should be continued in order to exclude relapse.

REFERENCES